

s il(w)18 and alzheimer?  
581352 IL  
1842927 18  
12821 IL(W)18  
267019 ALZHEIMER?  
S6 65 IL(W)18 AND ALZHEIMER?  
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S7 27 RD S6 (unique items)  
? t s7/7/all

7/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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0021279913 BIOSIS NO.: 200900621350  
Glyburide inhibits the Cryopyrin/Nalp3 inflammasome  
AUTHOR: Lamkanfi Mohamed; Mueller James L; Vitari Alberto C; Misaghi  
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JOURNAL: Journal of Cell Biology 187 (1): p61-70 OCT 5 2009 2009  
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ISSN: 0021-9525  
DOCUMENT TYPE: Article  
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ABSTRACT: Inflammasomes activate caspase-1 for processing and secretion of the cytokines interleukin-1 beta (IL-1 beta) and \*\*\*IL\*\*\* - \*\*\*18\*\*\*. Cryopyrin/NALP3/NLRP3 is an essential component of inflammasomes triggered by microbial ligands, danger-associated molecular patterns (DAMPs), and crystals. Inappropriate Cryopyrin activity has been incriminated in the pathogenesis of gouty arthritis, Alzheimer's, and silicosis. Therefore, inhibitors of the Nalp3 inflammasome offer considerable therapeutic promise. In this study, we show that the type 2 diabetes drug glyburide prevented activation of the Cryopyrin inflammasome. Glyburide's cyclohexylurea group, which binds to adenosine triphosphatase (ATP)-sensitive K<sup>+</sup> (K-ATP) channels for insulin secretion, is dispensable for inflammasome inhibition. Macrophages lacking K-ATP subunits or ATP-binding cassette transporters also activate the Cryopyrin inflammasome normally. Glyburide analogues inhibit ATP- but not hypothermia-induced IL-1 beta secretion from human monocytes expressing familial cold-associated autoinflammatory syndrome associated Cryopyrin mutations, thus suggesting that inhibition occurs upstream of Cryopyrin. Concurrent with the role of Cryopyrin in endotoxemia, glyburide significantly delays lipopolysaccharide-induced lethality in mice. Therefore, glyburide is the first identified compound to prevent Cryopyrin activation and microbial ligand-, DAMP-, and crystal-induced IL-1 beta secretion.

7/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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0021193754 BIOSIS NO.: 200900535191  
EFFECTS OF INTERLEUKIN-18 ON HUMAN SH-SY5Y NEUROBLASTOMA CELL LINE  
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JOURNAL: Journal of Neurochemistry 110 (Suppl. 2): p203 SEP 2009 2009  
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0020855691 BIOSIS NO.: 200900196025  
Interleukin-18 promoter polymorphisms and risk of late onset  
Alzheimer's disease  
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ITEM IDENTIFIER: doi:10.1016/j.brainres.2008.11.083  
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LANGUAGE: English

ABSTRACT: Pro- and anti-inflammatory cytokines play an important role in  
Alzheimer's disease (AD), and common polymorphisms of genes  
controlling their production have been shown to be associated with the  
susceptibility to sporadic AD. Interleukin ( \*\*\*IL\*\*\* )- \*\*\*18\*\*\* is a  
potent pro-inflammatory cytokine of the IL-1 superfamily, and increasing  
evidences indicate a crucial role for it in the pathogenesis of AD. To  
clarify the role of IL-18 as a potential cause for AD  
susceptibility, we investigated the effect of two functional  
polymorphisms in IL-18 promoter: -607 C/A (rs1946518) and  
-137 G/C (rs187238) for the risk of sporadic late onset Alzheimer's  
disease (LOAD) in a Han Chinese population of 109 patients and 109  
healthy controls matched for sex and age. All 218 subjects were also  
genotyped for the Apolipoprotein E (ApoE) polymorphisms. The results  
revealed that both -607 C allele and -137 G allele were associated with  
an increased risk of LOAD (odds ratios/OR=1.56, P=0.04, Power=0.96 and  
OR=1.85, P=0.03, Power=0.80, respectively), and these associations were  
influenced by the presence of ApoE epsilon 4 alleles. Moreover, they  
showed a highly significant synergistical interaction with the ApoE  
epsilon 4 allele (OR=5.70 and 4.64, respectively). Examination of the  
haplotypes identified the -607 C/-137 G haplotype to increase the risk of  
LOAD (OR=1.62, P=0.003, Power=0.97). These findings suggest that the  
functional polymorphisms in IL-18 promoter may be involved in  
the risk of developing sporadic LOAD in the Han Chinese population. (C)  
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7/7/4 (Item 4 from file: 5)  
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0020778926 BIOSIS NO.: 200900119260

Amyloid beta peptide promotes differentiation of pro-inflammatory human myeloid dendritic cells

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**ABSTRACT:** A key event of Alzheimer's disease (AD) pathogenesis is the production of amyloid beta peptides (A beta), which are hypothesized to lead to neurodegeneration by still unclear mechanisms, including a chronic inflammatory response characterized by innate immune cell activation and pro-inflammatory molecule release. Since dendritic cells (DCs) are central players of innate immune response and brain dendritic-like cells may have a crucial role in AD Pathogenesis, this study investigates the effects of A beta on human DC functions. Myeloid DCs differentiated in the presence of A beta 42 showed an increase in survival and soluble antigen uptake, a reduction in HLA molecule expression and in IL-10 and IL-12 production. Accordingly, A beta 42-treated DCs were impaired in inducing T cell proliferation and IL-2 production. On the other hand, A beta 42 treatment provided DCs with the ability to release higher levels of IL-1 beta, IL-6 and IL-18, than control DCs. These results demonstrate that A beta 42 can modulate the immune system by inducing pro-inflammatory DC differentiation, thus gaining new insights into AD pathogenesis and immune-based therapeutic intervention. (C) 2007 Elsevier Inc. All rights reserved.

7/7/5 (Item 5 from file: 5)

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0020778925 BIOSIS NO.: 200900119259

Expression of interleukin-18 is increased in the brains of Alzheimer 's disease patients

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LANGUAGE: English

**ABSTRACT:** The inflammatory cytokines can initiate nerve cell degeneration and enhance the plaque production typically found in Alzheimer's disease (AD). Interleukin-18 ( \*\*\*IL\*\*\* - \*\*\*18\*\*\* ) is an inflammatory cytokine, which can induce the expression of interferon-gamma. This interleukin shares similarities with the IL-1 family of proteins. Like IL-1 beta, IL-18 is cleaved by caspase-1 (ICE) to all active secreted form. We examined the expressions of \*\*\*IL\*\*\* - \*\*\*18\*\*\* , -1 beta and ICE in different brain regions from AD patients that were categorized with respect to the Braak stage, and age-matched with non-demented controls. The levels of total-RNA and protein of \*\*\*IL\*\*\* - \*\*\*18\*\*\* and ICE were increased, especially in the frontal lobe of AD patients and this change was not modified by ApoE genotype. Immunohistochemistry of AD brain samples detected IL-18 in microglia, astrocytes, and surprisingly in neurons, and it is also co-localized not only with amyloid-P plaques but also with tau. In CSF, elevated \*\*\*IL\*\*\* - \*\*\*18\*\*\* level was detected only in men and it also correlated with CSF tau ill MCI. \*\*\*IL\*\*\* - \*\*\*18\*\*\* may thus be a potential biomarker for men. Plasma levels of \*\*\*IL\*\*\* - \*\*\*18\*\*\* showed no correlation with the disease. In Conclusion, amyloid-P may induce the synthesis of IL-18, and IL-18 kinases involved in tau phosphorylation as a part of the amyloid-associated inflammatory reaction. (C) 2007 Elsevier Inc. All rights reserved.

7/7/6 (Item 6 from file: 5)  
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0020776679 BIOSIS NO.: 200900117013  
Interleukin-18 increases expression of kinases involved in tau phosphorylation in SH-SY5Y neuroblastoma cells  
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JOURNAL: Journal of Neuroimmunology 205 (1-2): p86-93 DEC 15 2008 2008  
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LANGUAGE: English

**ABSTRACT:** Inflammatory cytokines, produced mainly by activated microglia in the brain, can enhance neuronal degeneration and the amyloid-beta-plaque production involved in \*\*\*Alzheimer\*\*\* 's disease (AD). We previously demonstrated that the expression of the pro-inflammatory cytokine interleukin-18 (IL-18) colocalizes with plaques and hyperphosphorylated tau containing neurons in AD patients. Here we exposed neuron-like, differentiated SH-SY5Y neuroblastomas to IL-18 and observed that the protein levels of p35, Cdk5, GSK-3 beta, and Ser 15-phosphorylated p53 increased during 6 h-24 h. Tau phosphorylation and expression of cyclin G1, involved in neuronal regeneration, increased at 72 h. In vivo, over-expression of \*\*\*IL\*\*\* - \*\*\*18\*\*\* may induce hyperphosphorylation of tau and induce cell cycle activators. (C) 2008 Elsevier B.V. All rights reserved.

7/7/7 (Item 7 from file: 5)  
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0020701946 BIOSIS NO.: 200900042280  
Amyloid-beta oligomers set fire to inflammasomes and induce Alzheimer  
's pathology  
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JOURNAL: Journal of Cellular and Molecular Medicine 12 (6A): p2255-2262  
DEC 2008 2008  
ITEM IDENTIFIER: doi:10.1111/j.1582-4934.2008.00496.x  
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ABSTRACT: Genetic and molecular studies have confirmed the central role of amyloid-beta production and fibrillation in the pathogenesis of \*\*\*Alzheimer\*\*\* 's disease (AD). However, the pathological pathways from amyloid-beta peptide oligomerization to the major pathological hallmarks of AD, such as neurofibrillary tangles, inflammation and loss of cholinergic neurons, are largely unknown. The innate immunity defence system utilizes pattern recognition receptors to respond to a variety of danger- and pathogen-associated molecular structures. Amyloid-beta oligomers and fibrils and their cellular effects can activate the innate immunity defence and induce inflammatory and apoptotic responses in human brain. Amyloid-beta oligomers can interfere with many aspects of neuronal membrane functions and can evoke potassium (K<sup>+</sup>) efflux from neurons. A low K<sup>+</sup> concentration is a potent activator for the NALP1 inflammasomes, which then stimulate caspase-1 to cleave the proforms of IL-1 beta and \*\*\*IL-8\*\*\* - \*\*\*18\*\*\* cytokines. Interestingly, recent observations have demonstrated that amyloid-beta fibrils can activate NALP3 inflammasomes via the lysosomal damage in mouse microglia. We will review here the activation mechanisms of NALP inflammasomes in neurons and microglia and several downstream effects in brain demonstrating that toxic amyloid-beta oligomers and fibrils can light afire in inflammasomes and induce \*\*\*Alzheimer\*\*\* 's pathology.

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0020364040 BIOSIS NO.: 200800410979  
Interleukin-18 produced by peripheral blood cells is increased in Alzheimer's disease and correlates with cognitive impairment  
AUTHOR: Bossu Paola (Reprint); Ciaramella Antonio; Salani Francesca;  
Bizzoni Federica; Varsi Erika; Di Julio Fulvia; Giubilei Franco; Gianni  
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JOURNAL: Brain Behavior and Immunity 22 (4): p487-492 MAY 2008 2008  
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ABSTRACT: A body of evidence indicates that inflammation plays a pivotal role in AD pathogenesis. \*\*\*IL\*\*\* - \*\*\*18\*\*\* is a pro-inflammatory cytokine produced in the brain, emerging to be implicated in AD. Although no differences in circulating IL-18 levels were measured between AD patients and controls, a significant increased production of IL-18 was obtained from stimulated blood mononuclear cells of AD patients. This was true particularly in AD subjects carrying the C/C genotype at the -607 position of \*\*\*IL\*\*\* - \*\*\*18\*\*\* gene promoter. Furthermore, a significant correlation between IL-18 production and cognitive decline was observed in AD patients. Overall, these data indicate that IL-18-related inflammatory pathways, probably also in virtue of polymorphic IL-18 gene influence, are exacerbated in AD patients, and that this cytokine may indeed participate in pathogenic processes leading to dementia. (c) 2007 Elsevier Inc. All rights reserved.

7/7/9 (Item 9 from file: 5)  
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0020066544 BIOSIS NO.: 200800113483  
Pro-inflammatory cytokines and their effects in the dentate gyrus  
BOOK TITLE: Progress in Brain Research  
AUTHOR: Pickering Mark; O'Connor John J (Reprint)  
BOOK AUTHOR/EDITOR: Scharfman HE (Editor)  
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SERIES TITLE: PROGRESS IN BRAIN RESEARCH 163 p339-354 2007  
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BOOK PUBLISHER: ELSEVIER SCIENCE BV, SARA BURGERHARTSTRAAT 25, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS  
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ABSTRACT: The older notion of a central nervous system existing in essential isolation from the immune system has changed dramatically in recent years as the body of evidence relating to the interactions between these two systems has grown. Here we address the role of a particular subset of immune modulatory molecules, the pro-inflammatory cytokines, in regulating neuronal function and viability in the dentate gyrus of the hippocampus. These inflammatory mediators are known to be elevated in many neuropathological conditions, such as Alzheimer's disease, Parkinson's disease and ischaemic injury that follows stroke. Pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-alpha), interleukin 1-beta (IL-1 beta) and interleukin 18 (IL-18), have been shown to regulate neurotoxicity; although, due to the complexity of the cytokine action in neurons and glia, the effect may be either facilitatory or protective, depending on the circumstances. As well as their role in neurotoxicity and neuroprotection, the pro-inflammatory cytokines have also been shown to be potent regulators of synaptic function. In particular, TNF-alpha, IL-1 beta and \*\*\*IL\*\*\* - 18 have all been shown to inhibit long-term potentiation, a form of neuronal plasticity widely believed to underlie learning and memory, both in the early p38 mitogen activated protein kinase-dependant phase and the

later protein synthesis-dependant phase. In this article we address the mechanisms underlying these cytokine effects in the dentate gyrus of the hippocampus.

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0019991585 BIOSIS NO.: 200800038524  
Altered plasma cytokine levels in Alzheimer's disease: Correlation with the disease progression  
AUTHOR: Motta M; Imbesi R; Di Rosa M; Stivala F; Malaguarnera L (Reprint)  
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JOURNAL: Immunology Letters 114 (1): p46-51 NOV 30 2007 2007  
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ABSTRACT: Increasing evidence supports a propensity towards inflammation in \*\*\*Alzheimer\*\*\* 's disease (AD) pathogenesis. In our previous studies we observed high levels of IL-16, IL-18 and TGF-beta 1 mRNA expression in monocyte-macrophages of the peripheral blood of AD patients. The aim of this investigation was to determine the plasma levels of IL-12, IL-16, IL-18 and TGF-beta 1 in AD patients at different stages of the disease and to correlate the production of these cytokines with the disease progression. The levels of IL-12, IL-16, IL-18 and TGF-beta 1 resulted higher in AD-mild patients, were slightly lower in AD-moderate patients, whereas no significant difference was observed between AD-severe patients and non-demented age-matched subjects. The correlation values between cytokine plasma levels were dependent on the disease progression. Our data indicate that plasma levels of these inflammatory molecules follow the degree of AD suggesting a gradual decline of immune responsiveness in AD. (c) 2007 Elsevier B.V. All rights reserved.

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0019836473 BIOSIS NO.: 200700496214  
Interleukin 18 gene polymorphisms predict risk and outcome of Alzheimer's disease  
AUTHOR: Bossu Paola (Reprint); Ciaramella Antonio; Moro Maria Luisa; Bellincampi Lorenza; Bernardini Sergio; Federici Giorgio; Trequattrini Alberto; Macciardi Fabio; Spoletini Ilaria; Di Julio Fulvia; Caltagirone Carlo; Spalletta Gianfranco  
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JOURNAL: Journal of Neurology Neurosurgery & Psychiatry 78 (8): p807-811 AUG 2007 2007  
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ISSN: 0022-3050  
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LANGUAGE: English

**ABSTRACT:** Background and aim: Inflammation has been extensively implicated in the pathogenesis of \*\*\*Alzheimer\*\*\* 's disease ( AD). Although there is evidence of a key role for cytokines in neuroinflammation processes, so far the proinflammatory cytokine interleukin (IL)-18 has not been associated with AD. The aim of this study was to investigate the impact of two polymorphisms of the human IL-18 gene promoter at positions 2607 (C/A) and 2137 (G/C) on both susceptibility to and progression of AD. Results: The results revealed that the genotype distribution of the -607 (C/A) polymorphism was different between patients with AD and control subjects ( $\chi^2(2) = 7.99$ ,  $df = 2$ ,  $p = 0.0184$ ). In particular, carriers of the CC genotype were at increased risk of developing AD (OR 2.33; 95% CI 1.29 to 4.22;  $p = 0.0052$ ). The observed genotypes were in Hardy-Weinberg equilibrium, as for the 2607 polymorphism, whereas the 2137 polymorphism appeared in Hardy-Weinberg disequilibrium only in the patient group ( $p = 0.0061$ ). Finally, in a 2 year follow-up study, the 2137 CC genotype was strongly and specifically associated with a faster cognitive decline ( $F = 4.024$ ;  $df = 4, 192$ ;  $p = 0.0037$  for time by \*\*\*IL\*\*\* - \*\*\*18\*\*\* -137 G/C group interaction) with no interaction effect with the apolipoprotein E epsilon 4/non-epsilon 4 allele presence. Conclusion: As \*\*\*IL\*\*\* - \*\*\*18\*\*\* cytokine promoter gene polymorphisms have been previously described to have functional consequences on IL-18 expression, it is possible that individuals with a prevalent IL-18 gene variant have a dysregulated immune response, suggesting that IL-18 mediated immune mechanisms may play a crucial role in AD.

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16th Nippon-Medical-School-Foundation Academic Meeting for Foreign Researchers, Tokyo, JAPAN

AUTHOR: Anonymous  
JOURNAL: Journal of Nippon Medical School 73 (2): p106-111 APR 2006 2006  
CONFERENCE/MEETING: 16th Nippon-Medical-School-Foundation Academic Meeting for Foreign Researchers Tokyo, JAPAN 20051119,  
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19121861 BIOSIS NO.: 200600467256  
Chitotriosidase and inflammatory mediator levels in Alzheimer's disease and cerebrovascular dementia  
AUTHOR: Di Rosa Michelino; Dell'ombra Nicola; Zambito Anna Maria; Malaguarnera Mario; Nicoletti Ferdinando; Malaguarnera Lucia (Reprint)  
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JOURNAL: European Journal of Neuroscience 23 (10): p2648-2656 MAY 2006  
2006  
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LANGUAGE: English

**ABSTRACT:** Inflammation has been reported to be involved in the pathogenesis of cerebrovascular dementias (CvDs). This study investigated the involvement of Chitotriosidase (ChT), a chinolitic enzyme mainly produced by activated macrophages, in the pathophysiology of Alzheimer's disease (AD) and ischemic CvD. In addition, the levels of interleukin (IL)-16, IL-18, transforming growth factor (TGF) -beta 1 and superoxide anion (O-2(-)) were determined to evaluate the relationship between ChT levels, these cytokines and oxidative stress in both AD and ischemic CvD patients. The levels of ChT and IL-16, \*\*\*IL\*\*\* - \*\*\*18\*\*\*, and TGF-beta 1 mRNA were investigated using quantitative real-time polymerase chain reaction on macrophages of peripheral blood of 40 patients with AD, 40 patients with ischemic CvD and 40 non-demented age-matched subjects. The results show that ChT, IL-16 and O-2(-) levels significantly increased in ischemic CvD patients compared with AD patients and were significantly and positively correlated with IL-18\*\*\* and O-2(-). The production of \*\*\*IL\*\*\* - \*\*\*18\*\*\* was increased in both AD and ischemic CvD patients. TGF-beta 1 expression was higher in AD patients and was inversely correlated with the expression of ChT, IL-16 and \*\*\*IL\*\*\* - \*\*\*18\*\*\*, respectively. In non-demented age-matched subjects no significant changes in ChT and IL-16, IL-18, and TGF-beta 1 expression were found. Our results indicate that ChT, IL-16, \*\*\*IL\*\*\* - 18 and TGF-beta 1 are increased in ischemic CvD and AD, confirming that the immune system may play an important role in the development and progression of neurodegenerative disorders. In addition, the present findings suggest that ChT could also play a crucial role in pathological conditions such as CvD in which the inflammatory process is activated.

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18774921 BIOSIS NO.: 200600120316  
High cholesterol diet results in increased expression of interleukin-6 and caspase-1 in the brain of apolipoprotein E knockout and wild type mice  
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JOURNAL: Journal of Neuroimmunology 169 (1-2): p59-67 DEC 2005 2005  
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LANGUAGE: English

**ABSTRACT:** Inflammation in the central nervous System is an early hallmark of many neurodegenerative diseases including Alzheimer's disease (AD). Recently, increasing evidence Suggests that hypercholesterolemia during midlife and abnormalities in the cholesterol metabolism could have an important role ill the pathogenesis of AD. In the present Study, we have evaluated the effect of high cholesterol (HQ diet on the expression of interleukin-6 (IL-6), a cytokine involved in neurodegeneration, and caspase-1, that is responsible for the cleavage of the precursors of interleukin-1 beta (IL-1 beta) and interleukin-18 (IL-18) in the brain of apolipoprotein E (Apo E) knock-out (KO) and wild type (WT) mice. The density of IL-6-positive cells was increased in the hippocampus

(p<0.0001) and the dorsal part of the cortex (p<0.001) of KO and WT mice oil HC diet (KOHC and WTHC mice, respectively) compared to KO and WT mice oil ND (KOND and WTND mice, respectively). KOHC mice had increased caspase-1 positive cells and staining intensity in the hippocampus in comparison with WTHC mice (p<0.01). In the hippocampus, the density of caspase-1 positive cells was also higher in KOHC compared to KOND mice (p<0.05) and KOHC compared with WTHC mice (p<0.01). There was a major increase in caspase-1 immunoreactivity and cell density in both the dorsal part of the cortex (p<0.001) and the lateral part of the cortex (p<0.005) in KO and WT mice on HC diet compared to ND. The findings of the present study indicate that Chronic exposure to HC diet increases the expression of the two important inflammatory mediators IL-6 and caspase-1 in the brain of KO and WT mice. In the case of caspase-1, we report a major difference in the effect of HC diet on the KO mice compared to WT mice in the hippocampus. Increased expression of inflammatory mediators involved in neurodegeneration could be a potential mechanism by which hypercholesterolemia and HC diet increase the risk of AD. (C) 2005 Elsevier B.V. All rights reserved.

7/7/15 (Item 15 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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18387987 BIOSIS NO.: 200510082487  
Soluble interleukin-1 receptor type II, IL-18 and caspase-1 in  
mild cognitive impairment and severe Alzheimer's disease  
AUTHOR: Lindberg Catharina; Chromek Milan; Ahrengart Leif; Brauner Annelie;  
Schultzberg Marianne; Garlind Anita (Reprint)  
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JOURNAL: Neurochemistry International 46 (7): p551-557 JUN 05 2005  
ISSN: 0197-0186  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: In the present study, we have determined levels of soluble interleukin-1 (IL-1) receptor type II (sIL-1RII), interleukin-18 (IL-18) and caspase-1 in cerebrospinal fluid and serum from mild cognitive impairment patients that later progressed to \*\*\*Alzheimer\*\*\*'s disease (AD) and severe AD patients. Previous studies have shown that a chronic local inflammatory process is a part of AD neuropathology. In this process, activated microglial production of IL-1 seems to play an important role. In a previous study, we have shown increased levels of sIL-1RII in CSF from AD patients in a mild-moderate disease stage. In the present study, we found no significant differences in CSF or serum levels of sIL-1RII in either mild cognitive impairment or advanced AD patients as compared to control subjects. Likewise, there was no significant difference between mild cognitive impairment and severe AD patients. The same was true for caspase-1 and \*\*\*IL-1\*\*\* - \*\*\*IL-18\*\*\* serum levels, whereas CSF levels of caspase-1 and IL-18 were below detection limits. Our data indicate that the IL-1 system is relatively intact in the early and late stages of AD. (c) 2005 Elsevier Ltd. All rights reserved.

7/7/16 (Item 16 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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17762932 BIOSIS NO.: 200400143689  
P2X7 receptor modulation of beta-amyloid- and LPS-induced cytokine secretion from human macrophages and microglia.  
AUTHOR: Rampe David; Wang Lin; Ringheim Garth E (Reprint)  
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JOURNAL: Journal of Neuroimmunology 147 (1-2): p56-61 February 2004 2004  
MEDIUM: print  
ISSN: 0165-5728 \_(ISSN print)  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** To test whether extracellular ATP can play a role in the neuroimmunopathology of Alzheimer's disease (AD), we evaluated the capacity of the ATP-binding purinoreceptor, P2X7, to modulate cytokine secretion on cultured human macrophages and microglia pre-activated 24 h with the 42 amino acid beta-amyloid peptide (Abeta(1-42)) or lipopolysaccharide. Thirty minutes of exposure to the selective P2X7 agonist 2'-3'-O-(4-benzoylbenzoyl)adenosine 5'-triphosphate (BzATP) resulted in the secretion of IL-1beta after either Abeta(1-42) or LPS stimulation of human macrophages that was dependent on the concentration of the stimulus used to pre-activate the cells. Further tests on human microglia treated with BzATP (300  $\mu$ M) resulted in a 1.5- and 3.5-fold enhancement of IL-1alpha and IL-1beta secretion, respectively, from cells pre-activated by 10  $\mu$ M Abeta(1-42) and a 1.6- and 3.9-fold enhancement of IL-1alpha and IL-1beta secretion, respectively, from cells pre-activated by 1  $\mu$ g/ml LPS. BzATP induction of IL-1alpha and IL-1beta secretion from microglia was completely reversed by pre-incubation of the cells with the P2X7 antagonist, adenosine 5'-triphosphate 2',3'-acyclic dialcohol (oxidized ATP). In contrast to its effects on IL-1alpha and IL-1beta secretion, BzATP induced TNF-alpha after LPS stimulation, but not after stimulation with Abeta(1-42), induced IL-18 secretion regardless of whether microglia were pre-activated and attenuated IL-6 secretion after either LPS or Abeta(1-42) pre-activation. These results demonstrate that extracellular ATP can modulate Abeta-induced cytokine secretion from human macrophages and microglia and thus may play a role in the neuroimmunopathology of AD.

7/7/17 (Item 17 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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17516158 BIOSIS NO.: 200300484877  
Role of cytokines in neurological disorders.  
AUTHOR: Aarli Johan A (Reprint)  
AUTHOR ADDRESS: Department of Neurology, University of Bergen, Bergen, Norway\*\*Norway  
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JOURNAL: Current Medicinal Chemistry 10 (19): p1931-1937 October 2003 2003  
MEDIUM: print  
ISSN: 0929-8673  
DOCUMENT TYPE: Article; Editorial  
RECORD TYPE: Citation  
LANGUAGE: English

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17346301 BIOSIS NO.: 200300303790  
HIPPOCAMPAL INJURY AND GLIAL ACTIVATION WITHOUT UP - REGULATION OF  
INFLAMMATORY MEDIATORS: A GENE ARRAY ANALYSIS.  
AUTHOR: Little A R (Reprint); O'Callaghan J P (Reprint)  
AUTHOR ADDRESS: CDC-NIOSH, Morgantown, WV, USA\*\*USA  
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner  
2002 pAbstract No. 415.1 2002 2002  
MEDIUM: cd-rom  
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience  
Orlando, Florida, USA November 02-07, 2002; 20021102  
SPONSOR: Society for Neuroscience  
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Damage to the CNS results in a complex series of molecular and cellular events involving the injured cells and their supportive environment. Often, the response observed is complicated by factors extrinsic to CNS, such as an influx of blood-borne elements. We have used systemic administration of trimethyltin (TMT) (8.0 mg/kg) to damage the rat hippocampus without disrupting the blood brain barrier. This injury model causes extensive loss of hippocampal neurons and an accompanying microglial and astroglial activation, effects often linked to inflammatory responses (e.g. in \*\*\*Alzheimers\*\*\* disease). Previously, we have shown that TMT causes a marked and early up-regulation of the chemokine, MCP-1, in microglia. Surprisingly, expression of MCP-1 appeared to occur without an induction of other chemokines or proinflammatory cytokines. Thus, we now have used the Affymetrix gene chip as a more comprehensive approach for analysis of early gene expression patterns in the TMT injury model. At 2 and 5 days post TMT our findings confirm the induction of MCP-1 as well as a number of microglial (MHCII Ia antigen) and astroglial (GFAP, peripheral benzodiazepine receptors, glutamine synthetase, vimentin) genes. Genes implicated in the regulation of MCP-1 expression (p38 and JNK2 kinases), however, were not affected, whereas genes implicated in down stream effects of MCP-1 (ERK1 and PI3Kinase) were increased. Notably, genes associated with proinflammatory responses (e.g. TNF, IL-1, -2, -7, -9, -10, -12, -13, -15, -18, IFN, and complement) were not affected. These findings implicate a role for MCP-1 in hippocampal injury without the participation of other proinflammatory chemokines and cytokines.

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DIALOG(R)File 5:Biosis Previews(R)  
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16429347 BIOSIS NO.: 200200022858  
Modulation of cytokine release and pore formation by P2X7 channels in beta amyloid-activated human macrophages  
AUTHOR: Ringheim G E (Reprint); Wang L; Rampe D  
AUTHOR ADDRESS: Immunology, Aventis Pharmaceuticals, Inc, Bridgewater, NJ, USA\*\*USA  
JOURNAL: Society for Neuroscience Abstracts 27 (2): p2558 2001 2001  
MEDIUM: print  
CONFERENCE/MEETING: 31st Annual Meeting of the Society for Neuroscience  
San Diego, California, USA November 10-15, 2001; 20011110  
ISSN: 0190-5295  
DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** The P2Z/P2X7 receptor is a subtype of purinoreceptor expressed on the surface of macrophages and microglia, and which functions as an ATP ligand-gated ion channel. Expression of P2X7 can be upregulated by lipopolysaccharide (LPS), which in the presence of ATP, can enhance release of the cytokines IL-1beta and  $^{***}IL^{**} - ^{***}18^{***}$ . We investigated the effects of the P2X7 agonist 3'-O-(4-benzoylbenzoyl)-adenosine 5'-triphosphate (BzATP) on human peripheral blood-derived macrophages that had been activated for 5 h by the beta amyloid (1-42) peptide (10  $\mu$ M) implicated in the pathology of Alzheimer's disease, and we compared this to the effects of LPS. Exposure of these cells to BzATP lead to a concentration-dependent (1-1000  $\mu$ M) rapid release at 30 min of IL-1alpha IL-1beta, and  $^{***}IL^{**} - ^{***}18^{***}$ . At 300  $\mu$ M BzATP, respective increases were IL-1alpha (>five-fold), IL-1beta (>five-fold) and IL-1  $^{***}18^{***}$  (two-fold), but none for IL-6 or TNF-alpha. Cytokine release was blocked >80% by 300  $\mu$ M oxidized adenosine triphosphate (oxATP), a P2X7 antagonist. Treatment of the cells with oxATP 1 h before LPS or beta amyloid stimulation attenuated not only the extracellular release of cytokines, but also their intracellular accumulation over 24 h, suggesting an involvement of P2X7 receptors in cytokine gene expression as well as release. We propose that the P2X7 channels on activated macrophages and, by extension, microglia surrounding deposits of beta amyloid in the brains of Alzheimer's disease patients, are participating in the inflammatory processes leading to enhanced cytokine production.

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DIALOG(R)File 5:Biosis Previews(R)  
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15919731 BIOSIS NO.: 200100091570  
Beneficial effect(s) of n-3 fatty acids in cardiovascular diseases: But, why and how?  
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AUTHOR ADDRESS: EFA Sciences LLC, 1420 Providence Highway, Suite No. 266, Norwood, MA, 02062, USA\*\*USA  
JOURNAL: Prostaglandins Leukotrienes and Essential Fatty Acids 63 (6): p 351-362 December, 2000 2000  
MEDIUM: print  
ISSN: 0952-3278  
DOCUMENT TYPE: Article; Literature Review  
RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** Low rates of coronary heart disease was found in Greenland Eskimos and Japanese who are exposed to a diet rich in fish oil. Suggested mechanisms for this cardio-protective effect focused on the effects of n-3 fatty acids on eicosanoid metabolism, inflammation, beta oxidation, endothelial dysfunction, cytokine growth factors, and gene expression of adhesion molecules; But, none of these mechanisms could adequately explain the beneficial actions of n-3 fatty acids. One attractive suggestion is a direct cardiac effect of n-3 fatty acids on arrhythmogenesis. N-3 fatty acids can modify  $Na^+$  channels by directly binding to the channel proteins and thus, prevent ischemia-induced ventricular fibrillation and sudden cardiac death. Though this is an attractive explanation, there could be other actions as well. N-3 fatty acids can inhibit the synthesis and release of pro-inflammatory cytokines such as tumor necrosis factoralpha (TNFalpha) and interleukin-1 (IL-1)

and IL-2 that are released during the early course of ischemic heart disease. These cytokines decrease myocardial contractility and induce myocardial damage, enhance the production of free radicals, which can also suppress myocardial function. Further, n-3 fatty acids can increase parasympathetic tone leading to an increase in heart rate variability and thus, protect the myocardium against ventricular arrhythmias. Increased parasympathetic tone and acetylcholine, the principle vagal neurotransmitter, significantly attenuate the release of TNF, IL-1beta, IL-6 and \*\*\*IL\*\*\* - \*\*\*18\*\*\*. Exercise enhances parasympathetic tone, and the production of anti-inflammatory cytokine IL-10 which may explain the beneficial action of exercise in the prevention of cardiovascular diseases and diabetes mellitus. TNFalpha has neurotoxic actions, where as n-3 fatty acids are potent neuroprotectors and brain is rich in these fatty acids. Based on this, it is suggested that the principle mechanism of cardioprotective and neuroprotective action (s) of n-3 fatty acids can be due to the suppression of TNFalpha and IL synthesis and release, modulation of hypothalamic-pituitary-adrenal anti-inflammatory responses, and an increase in acetylcholine release, the vagal neurotransmitter. Thus, there appears to be a close interaction between the central nervous system, endocrine organs, cytokines, exercise, and dietary n-3 fatty acids. This may explain why these fatty acids could be of benefit in the management of conditions such as septicemia and septic shock, Alzheimer's disease, Parkinson's disease, inflammatory bowel diseases, diabetes mellitus, essential hypertension and atherosclerosis.

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DIALOG(R)File 5:Biosis Previews(R)  
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15587844 BIOSIS NO.: 200000306157  
Induction of cytokines in glial cells surrounding cortical beta-amyloid plaques in transgenic Tg2576 mice with Alzheimer pathology  
AUTHOR: Mehlihorn Gaby; Hollborn Margrit; Schliebs Reinhard (Reprint)  
AUTHOR ADDRESS: Department of Neurochemistry, Paul Flechsig Institute for Brain Research, University of Leipzig, Jahnallee 59, D-04109, Leipzig, Germany\*\*Germany  
JOURNAL: International Journal of Developmental Neuroscience 18 (4-5): p 423-431 July-August, 2000 2000  
MEDIUM: print  
ISSN: 0736-5748  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** beta-Amyloid plaque deposition observed in brains from Alzheimer patients, might function as immune stimulus for glial/macrophages activation, which is supported by observations of activated microglia expressing interleukin (IL)-1beta and elevated IL-6 immunoreactivity in close proximity to amyloid plaques. To elucidate the mechanisms involved in beta-amyloid-mediated inflammation, transgenic mice (Tg2576) expressing high levels of the Swedish double mutation of human amyloid precursor protein and progressively developing typical beta-amyloid plaques in cortical brain regions including gliosis and astrogliosis, were examined for the expression pattern of a number of cytokines. Using ribonuclease protection assay, interleukin (IL)-1alpha, -beta, IL-1 receptor antagonist, IL-6, IL-10, IL-12, IL-18, interferon-gamma, and macrophage migration inhibitory factor (MIF) mRNA were not induced in a number of cortical areas of Tg2576 mice regardless of the postnatal ages studied ranging between 2 and 13 months. Using immunocytochemistry for IL-1alpha,beta, IL-6, tumor necrosis factor

(TNF)-alpha, and macrophage chemotactic protein (MCP)-1, only IL-1beta was found to be induced in reactive astrocytes surrounding beta-amyloid deposits detected in 14-month-old Tg2576 mice. Using non-radioactive in situ hybridization glial fibrillary acidic protein (GFAP) mRNA was detected to be expressed by reactive astrocytes in close proximity to beta-amyloid plaques. The local immune response detected around cortical beta-amyloid deposits in transgenic Tg2576 mouse brain is seemingly different to that observed in brains from Alzheimer patients but may represent an initial event of chronic neuroinflammation at later stages of the disease.

7/7/22 (Item 1 from file: 73)  
DIALOG(R)File 73:EMBASE  
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0082365111 EMBASE No: 2008178850  
The diagnostic role of serum inflammatory and soluble proteins on dementia subtypes: Correlation with cognitive and functional decline  
Ozturk C.; Ozge A.; Yalim O.O.; Yilmaz I.A.; Delalioglu N.; Yildiz C.;  
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Behavioural Neurology ( Behav. Neurol. ) (Netherlands) December 1, 2007  
, 18/4 (207-215)  
CODEN: BNEUE ISSN: 0953-4180  
DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract  
LANGUAGE: English SUMMARY LANGUAGE: English  
NUMBER OF REFERENCES: 77

In the past years, the possible involvement of inflammation in the pathogenesis of dementia has been the subject of several investigations. However there are restricted data about the profile of the inflammatory and soluble proteins in well evaluated Alzheimer's disease (AD), vascular dementia (VD), mild cognitive impairment (MCI) and healthy controls. There are also no reliable data regarding the relationship between the overlapping protein levels and cognitive or functional decline. We measured levels of IL-1beta, IL-2, IL-6, IL-18, TNF-alpha, beta-Amyloid 1-40 and alpha SUB 1- antichymotrypsin levels in plasma in groups of total 82 subjects with AD, MCI, VD and controls using enzyme-linked immunosorbent assay (ELISA) method. Our study samples showed high levels of proinflammatory cytokine levels (especially IL-18) in all patient groups but only high levels of alpha SUB 1- antichymotrypsine in VD patients compared to controls. There is no significant correlation between the laboratory and clinical variables except for a link between IL-1beta and NPI scores of AD. In conclusion, this study yielded evidence of some shared mechanisms underlying AD and VD and thus motivates further studies of inflammatory markers in various types of dementia and MCI. (c) 2007 - IOS Press and the authors. All rights reserved.

7/7/23 (Item 2 from file: 73)  
DIALOG(R)File 73:EMBASE  
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0081725024 EMBASE No: 2007158831

The role of the purinergic P2X SUB 7 receptor in inflammation  
Lister M.F.; Sharkey J.; Sawatzky D.A.; Hodgkiss J.P.; Davidson D.J.;  
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eISSN: 1476-9255  
DOI: 10.1186/1476-9255-4-5  
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DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract  
LANGUAGE: English SUMMARY LANGUAGE: English  
NUMBER OF REFERENCES: 150

The inflammatory process, orchestrated against a variety of injurious stimuli, is composed of three inter-related phases; initiation, propagation and resolution. Understanding the interplay between these three phases and harnessing the beneficial properties of inflammation whilst preventing its damaging effects, will undoubtedly lead to the advent of much needed therapies, particularly in chronic disease states. The P2X SUB 7 receptor (P2X SUB 7R) is increasingly recognised as an important cell surface regulator of several key inflammatory molecules including IL-beta, \*\*\*IL\*\*\* - \*\*\*18\*\*\*, TNF-alpha and IL-6. Moreover, as P2X SUB 7R-dependent cytokine production is driven by activating the inflammasome, antagonists of this receptor are likely to have therapeutic potential as novel anti-inflammatory therapies. The function of the P2X SUB 7R in inflammation, immunity and its potential role in disease will be reviewed and discussed. (c) 2007 Lister et al; licensee BioMed Central Ltd.

7/7/24 (Item 3 from file: 73)  
DIALOG(R)File 73:EMBASE  
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0081284798 EMBASE No: 2006347155  
Interleukin-18 and transforming growth factor-beta 1 plasma levels in  
Alzheimer's disease and vascular dementia  
Malaguarnera L.; Motta M.; Di Rosa M.; Anzaldi M.; Malaguarnera M.  
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(307-312)  
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DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract  
LANGUAGE: English SUMMARY LANGUAGE: English  
NUMBER OF REFERENCES: 44

Inflammation has been involved in the development of dementia in cerebrovascular diseases. To investigate the cellular activation of the peripheral immune system in patients with Alzheimer's disease (AD) and vascular dementia (VaD), we determined the presence of IL-18 and TGF-beta1 in the plasma by using ELISA. The levels of IL-18 and TGF-beta1 were significantly elevated in patients with AD and VaD compared to non-demented, age-matched subjects. We found an inverse correlation between the levels of IL-18 and TGF-beta1 in AD patients. In VaD patients, the correlation between IL-18 and TGF-beta1 reached a borderline positive value. Whereas, in the non-demented, age-matched subjects, a positive correlation between IL-18 and TGF-beta1 levels was observed. These findings indicate that IL-18 and TGF-beta1 elevation is associated with AD and VaD patients, confirming that the immune system might exert a remarkable role in the development and progression of neurodegenerative disorders. Moreover, as different modifications were detected in the patients affected by AD and VaD, we propose that IL-18 and TGF-beta1 plasma levels might represent possible differential biomarkers. (c) 2006 Japanese Society of Neuropathology.

7/7/25 (Item 1 from file: 399)  
DIALOG(R)File 399:CA SEARCH(R)  
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151265386 CA: 151(12)265386y PATENT  
Compositions and methods for crystallizing antibody fragments  
INVENTOR(AUTHOR): Argiriadi, Maria A.; Borhani, David W.; Xiang, Tao; Wu, Chengbin; Ghayur, Tariq  
LOCATION: USA  
ASSIGNEE: Abbott Laboratories  
PATENT: PCT International ; WO 200999545 A1 DATE: 20090813  
APPLICATION: WO 200905568 (20090129) \*US 2008PV62887 (20080130) \*US 2008PV135739 (20080722)  
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A61K-0039/395 A I F B 20060101 H US  
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-related complex; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of Hypogammaglobulinemia...  
acquired hypogammaglobulinemia; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Pain...  
acute; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Respiratory distress syndrome...  
adult; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Cirrhosis...  
alc.; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Alcoholic liver disease...  
alc. cirrhosis; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Allergy... Eye,disease... Inflammation...  
allergic conjunctivitis; crystals of Fab fragments of antibodies to  
human interleukin-18 for treatment of  
Transplant and Transplantation... Transplant rejection...  
allograft; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Inflammation... Spinal column,disease...  
ankylosing spondylitis; crystals of Fab fragments of antibodies to  
human interleukin-18 for treatment of  
Transplant and Transplantation...  
allograft; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Inflammation... Spinal column,disease...  
ankylosing spondylitis; crystals of Fab fragments of antibodies to  
human interleukin-18 for treatment of  
Interleukin 18...  
antibody Fab fragment complexes; crystallization of  
Erythropoiesis...  
aplasia; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Anemia...  
aplastic; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Alopecia...  
areata; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Dermatitis...  
atopic; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Allergy...  
atopy; crystals of Fab fragments of antibodies to human interleukin-18  
for treatment of  
Autoimmune disease...  
autoimmune hemolytic anemia; crystals of Fab fragments of antibodies to  
human interleukin-18 for treatment of  
Anemia...  
autoimmune hemolytic; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Disease,animal...  
autoimmune lymphoproliferative syndrome; crystals of Fab fragments of  
antibodies to human interleukin-18 for treatment of  
Autoimmune disease... Inflammation... Thyroid gland,disease...  
autoimmune thyroiditis; crystals of Fab fragments of antibodies to  
human interleukin-18 for treatment of  
Autoimmune disease... Eye,disease... Inflammation...  
autoimmune uveitis; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Dermatitis... Hearing loss... Hypoglycemia... Hypothyroidism... Intestinal  
disease... Myocarditis... Thrombocytopenia... Thyroid gland,disease...  
autoimmune; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Eye,disease... Inflammation...  
blepharitis; crystals of Fab fragments of antibodies to human  
interleukin-18 for treatment of  
Bronchial disease... Obstructive pulmonary disease...

bronchiectasis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Skin,disease...  
bulous pemphigoid; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Skin,disease...  
bulous; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Spinal column,disease...  
cervical spondylosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Fatigue,biological...  
chronic fatigue syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Pain... Viral hepatitis...  
chronic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Eye,disease... Inflammation...  
conjunctivitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Addison's disease... Adrenal insufficiency... AIDS(disease)... Allergy...  
Alzheimer disease... Anaphylaxis... Antiphospholipid syndrome...  
Atherosclerosis... Autoimmune hepatitis... Autoimmune polyendocrinopathy...  
Behcet's syndrome... Bronchiolitis obliterans Cachexia... Cardiovascular disease... Celiac disease... Chlamydia... Cholestasis... Chronic obstructive pulmonary disease... Connective tissue disease... Crohn disease...  
Demyelination... Depression... Dermatomyositis... Drug toxicity...  
Endocarditis... Endometriosis... Fibrosis... Glomerulonephritis... Gout...  
Graves' disease... Hay fever... Heart failure... Hematopoietic neoplasm...  
Hepatitis B... Hepatitis C... Hyperthyroidism... Hypoparathyroidism...  
Inflammatory bowel disease... Ischemia... Kawasaki disease... Leukemia...  
Leukocytopenia... Liver disease... Lyme disease... Lymphoma... Mental and behavioral disorders... Multiple organ failure... Multiple sclerosis...  
Myasthenia gravis... Myelodysplastic syndromes... Myocardial infarction...  
Myocarditis... Neoplasm... Nephritis... Nephrotic syndrome...  
Osteoarthritis... Ovary,neoplasm... Pancreatitis... Paralysis... Parasitic infection... Parkinson's disease... Poliomyelitis... Prostate gland,neoplasm... Prostatitis... Pulmonary fibrosis... Rheumatic fever...  
Rheumatic heart disease... Sarcoidosis... Schizophrenia... Scleroderma...  
Sepsis... Shock(circulatory collapse)... Sjogren syndrome... Stroke...  
Systemic lupus erythematosis... Ulcerative colitis... Urticaria... Vascular restenosis... Vitiligo... Wound healing...  
crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Immune complexes...  
crystallization of antibody Fab fragment complexes with interleukin-18  
Human... Mouse... Mus musculus...  
crystallization of antibody Fab fragments targeting interleukin-18  
Disease,animal...  
dacryocystitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Retinal disease...  
diabetic retinopathy; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Cardiomyopathy...  
dilated; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Cutaneous lupus erythematosus...  
discoid; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of  
Reticuloendothelial system...

disease, histiocytosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Spinal column,disease...  
disk prolapse; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Blood coagulation disorders...  
disseminated intravascular coagulation; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Eye,disease...  
dry eye syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Eye,disease... Inflammation...  
endophthalmitis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Pneumonia...  
eosinophilic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Analgesics... Anti-AIDS agents...  
Fab fragments of antibodies to human interleukin-18

Ovarian disease...  
failure, premature, autoimmune; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Fertility disorders...  
female; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Polyoxylalkylenes...  
for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

Protein sequences...  
for heavy chain fragments and light chains of human and mouse antibodies to interleukin-18

Antibodies and Immunoglobulins...  
fragments, Fab; crystallization of antibody Fab fragments targeting interleukin-18

Arteritis...  
giant cell arteritis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Kidney disease...  
Goodpasture syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Transplant and Transplantation...  
graft-vs.-host reaction; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Nervous system,disease...  
Guillain-Barre syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Anemia...  
hemolytic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Purpura(disease)...  
Henoch-Schoenlein; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Injury...  
hepatic, alc.; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Disease,animal...  
histiocytosis; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Disease,animal...  
Hughes Syndrome; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Nervous system,disease...

Huntington's chorea; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Thrombocytopenia...

idiopathic; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Antibodies and Immunoglobulins...

IgG1, monoclonal; crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes of

Myositis...

inclusion body; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Streptococcus...

infection; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Liver disease...

injury, alc.; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

Diabetes mellitus...

insulin-dependent; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

CAS REGISTRY NUMBERS:

1180118-79-9 1180118-80-2 1180118-81-3 1180118-82-4 1180118-83-5

1180118-84-6 amino acid sequence; crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

64-19-7 77-92-9 288-32-4 7664-38-2 biological studies, buffer; for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

7786-30-3 biological studies, for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

9004-10-8 biological studies, resistance; crystals of Fab fragments of antibodies to human interleukin-18 for treatment of

103-47-9 150-25-4 1132-61-2 1135-40-6 4432-31-9 6976-37-0 15132-04-4 buffer; for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

1179822-04-8 1179822-05-9 1179822-06-0 1179822-07-1 crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

77-86-1 7365-45-9 15471-17-7 25322-68-3 87993-44-0 for crystallization of anti-interleukin 18 antibody Fab fragments and IL-18/Fab complexes

7/7/26 (Item 2 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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143323906 CA: 143(18)323906r CONFERENCE PROCEEDING

CAMP-specific Pde4B mediated AB-induced microglial activation

AUTHOR(S): Sebastiani, G.; Morissette, C.; Lagace, C.; Boule, M.;

Ouellette, M. J.; McLaughlin, R. W.; Lacombe, D.; Gervais, F.; Tremblay, P.

LOCATION: Neurochem (International) Limited, Laval, QC, Can., H7V 4A7

JOURNAL: Amyloid Amyloidosis, (Int. Symp. Amyloidosis), 10th (Amyloid and Amyloidosis, (International Symposium on Amyloidosis), 10th, Tours, France, Apr. 18-22, 2004) EDITOR: Grateau, Gilles (Ed), Kyle, Robert A. (Ed), Skinner, Martha (Ed), DATE: 2005 PAGES: 382-384 CODEN: 69GPWW

LANGUAGE: English MEETING DATE: 20040000 PUBLISHER: CRC Press LLC, Boca Raton, Fla

SECTION:

CA214010 Mammalian Pathological Biochemistry

IDENTIFIERS: amyloidbeta microglia cAMP phosphodiesterase 4B gene

Alzheimer, cDNA array technol

DESCRIPTORS:

Neuroglia...

microglia; soluble and fibrillary amyloid- $\beta$ 1-42 induced microglia cell activation upregulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.

Gene expression profiles,animal... DNA microarray technology...

s- and f $\text{A}\beta$ 1-42 induced microglia cell activation upregulated Pde4B, MIP-2, Lyn, Kcnk3, Scrab1, PTP1B, IL-1 $\alpha$ , IL-18, M-ras, Nr2c2, Gsr, Slc5a2, Inpp4a, Fabp5, TNF- $\alpha$ , IP-10 gene express

Gene,animal... Human... Alzheimer's disease...

soluble and fibrillary amyloid- $\beta$ 1-42 induced microglia cell activation up-regulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.

CAS REGISTRY NUMBERS:

9036-21-9 60-92-4 107761-42-2 soluble and fibrillary amyloid- $\beta$ 1-42 induced microglia cell activation up-regulated cAMP-specific phosphodiesterase 4B gene expression as shown by using cDNA array technol.

7/7/27 (Item 3 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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142462257 CA: 142(25)462257x PATENT

Human antibodies to interleukin-18

INVENTOR(AUTHOR): Ghayur, Tariq; Labkovsky, Boris; Voss, Jeffrey W.; Green, Larry; Babcock, John; Jia, Xiao-chi; Wieler, James; Kang, Jaspal Singh; Hedberg, Brad

LOCATION: USA

PATENT: U.S. Pat. Appl. Publ. ; US 20050100965 A1 DATE: 20050512

APPLICATION: US 20031706689 (20031112)

PAGES: 87 pp. CODEN: USXXCO LANGUAGE: English

PATENT CLASSIFICATIONS:

CLASS: 435007100; G01N-033/53A; C07H-021/04B; C07K-016/24B;

C12N-005/06B

SECTION:

CA215001 Immunochemistry

CA201XXX Pharmacology

CA214XXX Mammalian Pathological Biochemistry

CA263XXX Pharmaceuticals

IDENTIFIERS: interleukin 18 human antibody

DESCRIPTORS:

Skin,disease...

acanthosis nigra; in combination therapy with human antibodies to interleukin-18

Antibodies and Immunoglobulins...

acquired hypogammaglobulinemia; in combination therapy with human antibodies to interleukin-18

Respiratory distress syndrome...

adult; in combination therapy with human antibodies to interleukin-18

Cirrhosis...

alc.; in combination therapy with human antibodies to interleukin-18

Lung,disease...

alveolitis, cryptogenic fibrosing; in combination therapy with human antibodies to interleukin-18

Spinal column,disease... Inflammation...

ankylosing spondylitis; in combination therapy with human antibodies to interleukin-18

Artery,disease... Inflammation...

arteritis, giant cell; in combination therapy with human antibodies to interleukin-18

Artery,disease... Inflammation...  
arteritis, Takayasu's disease; in combination therapy with human antibodies to interleukin-18

Disease,animal...  
arthropathy; in combination therapy with human antibodies to interleukin-18

Allergy...  
atopy; in combination therapy with human antibodies to interleukin-18

Hepatitis...  
autoimmune, cryptogenic; in combination therapy with human antibodies to interleukin-18

Anemia(disease)... Autoimmune disease...  
autoimmune hemolytic anemia; in combination therapy with human antibodies to interleukin-18

Autoimmune disease...  
autoimmune thrombocytopenia; in combination therapy with human antibodies to interleukin-18

Thyroid gland,disease... Autoimmune disease... Inflammation...  
autoimmune thyroiditis; in combination therapy with human antibodies to interleukin-18

Hypothyroidism...  
autoimmune; in combination therapy with human antibodies to interleukin-18

Hepatitis...  
B; in combination therapy with human antibodies to interleukin-18

Bronchi,disease... Inflammation...  
bronchiolitis; in combination therapy with human antibodies to interleukin-18

Hepatitis...  
C; in combination therapy with human antibodies to interleukin-18

Mycosis...  
candidiasis, mucocutaneous; in combination therapy with human antibodies to interleukin-18

Antibodies and Immunoglobulins...  
chimeric; to human interleukin-18

Animal cell line...  
CHO; for preparation of antibodies to human interleukin-18

Biliary tract,disease...  
cholestasis; in combination therapy with human antibodies to interleukin-18

Fatigue,biological...  
chronic fatigue syndrome; in combination therapy with human antibodies to interleukin-18

Radionuclides,biological studies... Fluorescent substances... Luminescent substances...  
conjugates with antibodies to human interleukin-18

Enzymes,biological studies...  
conjugates; with antibodies to human interleukin-18

Animal cell line...  
COS; for preparation of antibodies to human interleukin-18

Inflammation...  
Crohn's disease; in combination therapy with human antibodies to interleukin-18

Intestine,disease...  
Crohn's; in combination therapy with human antibodies to interleukin-18

Mental disorder...  
depression; in combination therapy with human antibodies to interleukin-18

Peptides,biological studies...  
depsipeptides, polymeric; for delivery of antibodies to human interleukin-18

Heart,disease...  
dilated cardiomyopathy; in combination therapy with human antibodies to interleukin-18

Lupus erythematosus...  
discoid; in combination therapy with human antibodies to interleukin-18

Platelet(blood)...  
disease, autoimmune thrombocytopenia; in combination therapy with human antibodies to interleukin-18

Platelet(blood)...  
disease, thrombocytopenia, idiopathic; in combination therapy with human antibodies to interleukin-18

Joint,anatomical...  
disease; in combination therapy with human antibodies to interleukin-18

Blood coagulation...  
disseminated intravascular; in combination therapy with human antibodies to interleukin-18

Lung,disease...  
eosinophilia; in combination therapy with human antibodies to interleukin-18

Heart,disease... Ovary,disease...  
failure; in combination therapy with human antibodies to interleukin-18

Liver,disease...  
fatty; in combination therapy with human antibodies to interleukin-18

Fertility...  
female, disorder; in combination therapy with human antibodies to interleukin-18

Lung,disease...  
fibrosis; in combination therapy with human antibodies to interleukin-18

Plasmid vectors... Drug delivery systems...  
for antibodies to human interleukin-18

Polyanhydrides... Polyesters,biological studies... Albumins,biological studies... Collagens,biological studies... Fibrins... Gelatins,biological studies... Oligosaccharides,biological studies...

Glycosaminoglycans,biological studies... Polyoxyalkylenes,biological studies...  
for delivery of antibodies to human interleukin-18

Protein sequences...  
for human antibody variable regions to interleukin-18

Escherichia coli... Saccharomyces cerevisiae...  
for preparation of antibodies to human interleukin-18

Antibodies and Immunoglobulins...  
fragments; to human interleukin-18

Kidney,disease... Inflammation...  
glomerulonephritis; in combination therapy with human antibodies to interleukin-18

Kidney,disease...  
Goodpasture's syndrome; in combination therapy with human antibodies to interleukin-18

Transplant and Transplantation...  
graft-vs.-host reaction; in combination therapy with human antibodies to interleukin-18

T cell(lymphocyte)...  
helper cell; neutralizing antibodies to human interleukin-18 modulate function of

Anemia(disease)...  
hemolytic; in combination therapy with human antibodies to interleukin-18

Purpura(disease)...  
Henoch-Schoenlein's; in combination therapy with human antibodies to interleukin-18

Infection...  
hepatitis B; in combination therapy with human antibodies to interleukin-18

Infection...  
hepatitis C; in combination therapy with human antibodies to interleukin-18

Immunotherapy...  
human antibodies to interleukin-18

Antibodies and Immunoglobulins...  
humanized; to human interleukin-18

Nervous system,disease...  
Huntington's chorea; in combination therapy with human antibodies to interleukin-18

Blood,disease...  
idiopathic thrombocytopenia; in combination therapy with human antibodies to interleukin-18

Leukocytopenia...  
idiopathic; in combination therapy with human antibodies to interleukin-18

Antibodies and Immunoglobulins...  
IgA, monoclonal; to human interleukin-18

Antibodies and Immunoglobulins...  
IgG1, monoclonal; to human interleukin-18

Antibodies and Immunoglobulins...  
IgG2, monoclonal; to human interleukin-18

Antibodies and Immunoglobulins...  
IgG3, monoclonal; to human interleukin-18

Antibodies and Immunoglobulins...  
IgM, monoclonal; to human interleukin-18

CAS REGISTRY NUMBERS:

851493-81-7 851493-82-8 851493-83-9 851493-84-0 851493-46-4  
851493-47-5 851493-48-6 851493-49-7 851493-50-0 851493-51-1  
851493-52-2 851493-53-3 851493-54-4 851493-55-5 851493-56-6  
851493-57-7 851493-58-8 851493-59-9 851493-60-2 851493-61-3  
851493-62-4 851493-63-5 851493-64-6 851493-65-7 851493-66-8  
851493-67-9 851493-68-0 851493-69-1 851493-70-4 851493-71-5  
851493-72-6 851493-73-7 851493-74-8 851493-75-9 851493-76-0  
851493-77-1 851493-78-2 851493-79-3 851493-80-6 amino acid  
sequence; preparation and characterization of human antibodies to interleukin-18

384463-34-7 385172-72-5 391765-35-8 384440-08-8 384557-61-3  
391783-87-2 152649-01-9 391528-67-9 171212-01-4 384581-28-6  
391777-17-6 384655-19-0 389406-79-5 176145-94-1 389184-77-4  
175113-26-5 391551-76-1 391535-51-6 384735-80-2 392193-44-1  
391815-18-2 140735-36-0 184342-40-3 140025-63-4 161273-84-3  
391525-76-1 389182-06-3 140741-74-8 382730-07-6 139840-61-2  
389195-33-9 182940-44-9 150326-43-5 176348-12-2 384497-75-0  
175299-02-2 187859-73-0 384438-97-5 384442-87-9 389182-22-3  
384515-94-0 391546-79-5 391530-32-8 140062-81-3 180604-89-1  
140049-62-3 188329-74-0 391783-55-4 391847-57-7 168523-60-2  
175196-96-0 140278-85-9 156678-90-9 163953-68-2 391536-35-9  
151658-42-3 147351-39-1 150510-74-0 384728-18-1 160475-87-6  
184941-12-6 140509-06-4 392193-49-6 141010-21-1 392204-67-0  
141010-19-7 141705-88-6 391524-99-5 391766-17-9 152347-85-8  
140539-49-7 144532-01-4 172712-00-4 391528-24-8 175524-88-6  
384976-62-9 384463-75-6 391772-01-3 384463-84-7 398425-27-9  
391525-11-4 140281-74-9 140740-20-1 391527-32-5 391548-59-7  
384463-53-0 389193-51-5 164952-58-3 391550-27-9 160075-32-1  
384463-99-4 384440-42-0 384440-01-1 391524-76-8 181828-95-5  
391806-64-7 391834-40-5 140746-12-9 398114-34-6 151973-86-3  
384480-59-5 178734-25-3 384528-75-0 157417-40-8 391540-54-8

391523-44-7 168042-97-5 391523-63-0 391536-91-7 391528-57-7  
391838-61-2 389357-20-4 389200-61-7 173005-29-3 183984-01-2  
140033-47-2 190920-95-7 183819-07-0 384536-34-9 384506-42-7  
184632-60-8 165149-73-5 384653-03-6 140098-98-2 391540-52-6  
139810-14-3 384488-43-1 169733-38-4 189023-52-7 391548-28-0  
384452-11-3 177929-43-0 391831-79-1 390187-88-9 392215-20-2  
391535-10-7 164952-76-5 384527-97-3 391831-70-2 174128-16-6  
389310-50-3 149737-37-1 391529-34-3 140576-31-4 391791-22-3  
140961-11-1 391818-98-7 141165-70-0 180173-20-0 384520-60-9  
189463-46-5 160900-81-2 391544-82-4 155117-74-1 140316-52-5  
391557-48-5 152371-82-9 384492-73-3 391758-90-0 140030-59-7  
142693-77-4 391561-76-5 391534-20-6 391532-21-1 168605-03-6  
384655-55-4 391558-60-4 389415-01-4 252793-80-9 391839-12-6  
150861-19-1 389406-78-4 389192-44-3 183983-99-5 186445-92-1  
391547-30-1 183983-98-4 226451-68-9 398095-28-8 391532-62-0  
384975-98-8 140331-48-2 183469-53-6 antibodies to human  
interleukin-18 for modulation of IL-18-responsive genes  
9004-34-6 biological studies, for delivery of antibodies to human  
interleukin-18  
57-50-1 biological studies, in delivery of antibodies to human  
interleukin-18  
351881-10-2 851221-25-5 851221-27-7 851221-29-9 851221-31-3  
851221-33-5 851221-35-7 384331-78-6 851221-38-0 851221-40-4  
851221-42-6 851221-44-8 851221-46-0 851221-48-2 851221-49-3  
851221-50-6 851221-51-7 851221-52-8 851221-53-9 851221-54-0  
851221-55-1 851221-56-2 701915-28-8 851221-57-3 220541-02-6  
851221-58-4 851221-59-5 851221-60-8 141977-02-8 694439-07-1  
851221-61-9 706781-57-9 851221-62-0 851221-63-1 851221-64-2  
851221-65-3 851221-66-4 328897-77-4 851221-67-5 851221-68-6  
220540-59-0 851221-69-7 851221-70-0 851221-71-1 851221-72-2  
851221-73-3 851221-74-4 851221-75-5 705288-83-1 851221-76-6  
851221-77-7 851221-78-8 851221-79-9 851221-80-2 851221-81-3  
851221-82-4 328897-74-1 851221-83-5 851221-84-6 851221-85-7  
851221-86-8 851221-87-9 851221-88-0 851221-89-1 851221-90-4  
851221-91-5 851221-92-6 851221-93-7 280106-92-5 851221-94-8  
384331-70-8 851221-95-9 851221-96-0 851221-97-1 217478-43-8  
851221-98-2 851221-99-3 851222-00-9 851222-01-0 848074-72-6  
851222-02-1 851222-03-2 851222-04-3 851222-05-4 851222-06-5  
851222-07-6 851222-08-7 173480-65-4 173480-66-5 851222-09-8 CDR  
regions of human antibodies to interleukin-18  
14762-75-5D 15117-53-0D 10098-91-6D 14133-76-7D 15750-15-9D  
14158-31-7D 10043-66-0D 14265-75-9D 13967-65-2D 15766-00-4D  
conjugates, uses, with antibodies to human interleukin-18  
58-85-5D 10028-17-8D conjugates, with antibodies to human interleukin-18  
108-31-6D copolymers with alkyl vinyl ethers, for delivery of antibodies  
to human interleukin-18  
9004-34-6D derivs., for delivery of antibodies to human interleukin-18  
9003-01-4 75268-90-5 26100-51-6 34346-01-5 26063-00-3 25248-42-4  
31621-87-1 25322-68-3 40704-75-4 9002-89-5 9003-39-8 9005-32-7  
9004-61-9 26023-30-3 24980-41-4 for delivery of antibodies to human  
interleukin-18  
7585-39-9D hydroxypropyl ethers, in delivery of antibodies to human  
interleukin-18  
59-05-2 104987-11-3 59865-13-3 53123-88-9 in combination therapy with  
human antibodies to interleukin-18  
99-20-7 585-86-4 9004-74-4 in delivery of antibodies to human  
interleukin-18  
691397-13-4D polyols, for delivery of antibodies to human interleukin-18  
851496-80-5 unclaimed protein sequence; human antibodies to interleukin-18  
?